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The early events of oxygen and glucose deprivation: setting the scene for neuronal death?

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It is generally thought that neuronal death caused by a reduction in oxygen or glucose supply, or both, occurs as a result of massive increases in the extracellular concentrations of excitatory amino acid neurotransmitters, particularly glutamate. A pertinent question is what happens before this increase, because measures which prevent extracellular accumulation of glutamate could have potential for clinical use in, for example, management of acute stroke. This article will review the major pathophysiological responses which occur up until the time of accumulation of glutamate. Withdrawal of energy substrate quickly leads to modest changes in membrane potential and intracellular and extracellular ion concentrations. Depression of action-potential-dependent synaptic transmission occurs a little later and might, in part, reflect actions of adenosine. Increases in the extracellular concentration of excitatory amino acids to neurotoxic levels take place only as membrane potential falls rapidly towards 0mV, coincident with massive changes in ion gradients.

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